SUDDEN GAINS DURING COGNITIVE-BEHAVIORAL TREATMENT OF OBSESSIVE-COMPULSIVE DISORDER

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ABSTRACT

Jennifer L. Buchholz: Sudden Gains During Cognitive-Behavioral Treatment of Obsessive-Compulsive Disorder
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A sudden gain is defined as a decrease in symptom severity between two consecutive treatment sessions that is large (a) in absolute terms, (b) compared to severity before the gain, and (c) compared to fluctuations before and after the gain. Although research documents a link between sudden gains and treatment for depression and anxiety, findings in the context of obsessive-compulsive disorder (OCD) treatment are mixed. The present study investigated the relationship between sudden gains and treatment outcome in 44 adults with OCD by measuring OCD symptoms dimensionally and comparing individuals who experience sudden gains to those who experience gradual gains of similar magnitude. Sudden gains were observed among 27% of participants, with highest rates among individuals with primary contamination symptoms. Participants who experienced a sudden gain had greater OCD symptom reductions at post-treatment (but not at follow-up), and this difference did not persist after controlling for gain magnitude.

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INTRODUCTION

Obsessive-compulsive disorder (OCD) is among the most common and functionally impairing psychological conditions, with a lifetime prevalence rate of 1% to 3% in the general population (Adam, Meinlschmidt, Gloster, & Lieb, 2012; Ruscio, Stein, Chiu, & Kessler, 2010). Evidence from numerous studies supports the efficacy and effectiveness of cognitive-behavioral therapy (CBT), with an emphasis on exposure and response prevention (ERP), as the first-line treatment of OCD (Olatunji, Davis, Powers, & Smits, 2013). ERP involves repeated confrontation with internal and external obsessional cues without the use of compulsive rituals. This intervention has demonstrated superiority to other psychological interventions (Lindsay, Crino, & Andrews, 1997) and medication (Foa et al., 2005) for OCD, and the majority of individuals who complete ERP experience a reduction in symptoms (Abramowitz, Taylor, & McKay, 2009). However, about 20-30% of individuals who receive ERP are considered treatment non-responders and continue to experience significant impairment (Foa & Kozak, 1996; Schruers, Koning, Luermans, Haack, & Griez, 2005). Identifying predictors of treatment outcome may help clinicians adapt interventions to enhance the effectiveness of ERP.

One potential prognostic indicator is a *sudden gain*, defined as a large, rapid, and stable decrease in symptoms between treatment sessions. Tang and DeRubeis (1999), studying CBT for depression, were the first to report a relationship between sudden gains and treatment response. They quantified a sudden gain as a symptom reduction between two consecutive therapy sessions that is large (a) in absolute terms, (b) compared to severity before the gain, and (c) compared to fluctuations before and after the gain. They identified sudden gains in more than 50% of patients

receiving cognitive-behavioral treatment for depression, and the average gain magnitude was 11 points on the Beck Depression Inventory (BDI; Beck & Steer, 1987). Tang and DeRubeis found that sudden gains predicted treatment outcome, such that individuals who experienced a sudden gain had greater overall depressive symptom reduction from pre- to post-treatment than did those who did not show a sudden gain. Using similar criteria, researchers have replicated these findings in the treatment of depression (e.g. Hardy et al., 2005) as well as social anxiety disorder (Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006), generalized anxiety disorder (Present et al., 2008), panic disorder (Clerkin, Teachman, & Smith-Janik, 2008), and posttraumatic stress disorder (Doane, Feeny, & Zoellner, 2010). A recent meta-analysis of sudden gains during treatment for anxiety and depression found a medium effect for sudden gains as predictors of outcome (Hedge's g = 6.2, SE = .09), with greater effect sizes for CBT than other therapies (Aderka, Nickerson, Bøe, & Hofmann, 2012).

Only two studies to date have examined sudden gains during the treatment of OCD. Combining data from two randomized controlled trials, Aderka and colleagues (2012) found sudden gains in 34.1% of 91 individuals receiving cognitive therapy, exposure therapy, and both psychological treatments in combination with fluvoxamine. In this study, individuals who experienced sudden gains reported fewer OCD symptoms following treatment relative to those who did not experience sudden gains, and this difference was maintained at follow-up. Seeking to replicate and extend these findings, Collins and Coles (2017) examined sudden gains among 27 patients undergoing ERP for OCD. Fifty-two percent of patients in their sample experienced a sudden gain, but the occurrence of a sudden gain was not significantly associated with OCD symptom reduction. That is, contrary to hypotheses, individuals who experienced a sudden gain did not experience a greater decrease in OCD symptoms (pre-to post-treatment) than those

without a sudden gain. Given these mixed results, additional research is warranted to examine the relationship between sudden gains and symptom reduction among individuals receiving treatment for OCD. Accordingly, the present study aimed to extend the scope of findings on sudden gains during ERP treatment of OCD.

One important limitation of both previous sudden gain studies in OCD is that they measured improvement in *global* OCD symptoms. Findings from numerous structural analyses of OCD symptoms suggest, however, that the substantial heterogeneity of OCD distills into four symptom dimensions: (a) contamination obsessions and de-contamination rituals, (b) obsessions about responsibility for harm and checking rituals, (c) unacceptable obsessional thoughts and mental neutralizing and reassurance-seeking rituals, and (d) incompleteness obsessions and order/symmetry rituals (for a review see McKay et al., 2004). Moreover, differences in response to ERP have been identified across these dimensions. Individuals with symptoms related to contamination tend to fare better in ERP relative to other presentations of OCD, whereas those with symptoms related to unacceptable thoughts are more likely to have attenuated outcomes (Kelley, Storch, Merlo, & Geffken, 2008). Accordingly, sudden gains in dimensional symptoms may provide more precise and clinically useful information than sudden gains in global symptoms. We therefore measured OCD symptoms dimensionally in the present study, which allowed us to examine relationships between sudden gains and OCD symptom presentation in a novel way.

Another important limitation of previous studies is that they did not directly compare the effects of a sudden gain to the effects of a gradual gain of similar magnitude. That is, in the majority of sudden gains studies, groups of patients who experienced sudden gains were compared to heterogeneous groups of all participants of the same sample who did not experience

sudden gains (e.g., Tang & DeRubeis, 1999; Aderka et al., 2012, Collins & Coles, 2017). As Greenfield, Gunthert, and Haaga (2011) have pointed out, such comparison groups may include individuals who (a) experienced worsening symptoms, (b) did not improve, and/or (c) improved gradually during treatment. Therefore, findings in previous studies suggesting a relationship between sudden gains and treatment outcome may actually reflect the importance of *substantial* gains during treatment, rather than *sudden* gains in particular. Greenfield, Gunthert, and Haaga argue that more meaningful comparisons are between (a) individuals who experience sudden gains and (b) those who experience similarly sizable, but *gradual*, improvement during treatment. They measured overall therapy outcomes for "sudden gainers" and "gradual gainers" in a psychotherapy training clinic, and found that outcomes were significantly better for "sudden gainers." Accordingly, we extended existing research in OCD by comparing individuals who experienced sudden gains to those who experienced gradual gains of similar magnitude.

It is also important to consider changes in psychological constructs that may be associated with rapid reductions in symptomatology. In their seminal study, for example, Tang and DeRubeis (1999) found that changes in cognitions (i.e., positive shifts in thinking patterns) preceded sudden gains in depressive symptoms, a finding that was later replicated by Tang, DeRubeis, Beberman, and Pham (2005). Applying this framework to anxiety disorders, Norton, Klenck, and Barrera (2010) found greater cognitive changes in individuals who experienced sudden gains relative to those who did not during cognitive-behavioral group therapy. However, investigators who have sought to replicate these findings have not always found sudden gains to be associated with cognitive change (e.g., Hofmann et al., 2006; Kelly, Roberts, & Ciesla, 2005).

The relationship between cognitive changes and sudden gains during ERP for OCD has not yet been examined. ERP is thought to involve a cognitive mechanism of change whereby

exposure to feared stimuli alters expectancies about the likelihood and severity of threat, which leads to behavioral change (e.g., Craske et al., 2008; Jacoby & Abramowitz, 2016). According to cognitive-behavioral conceptualizations of OCD (e.g., Salkovskis, 1996), obsessions arise from the cognitive misappraisal of normal, universally occurring intrusive thoughts as highly threatening (e.g., "thinking of harming a baby means I am a dangerous person"). Accompanying compulsions and rituals are performed with the aim of controlling such thoughts and reducing related distress. Rituals are negatively reinforced by the short-term relief from obsessional anxiety that the engender, but also maintain obsessional fear by preventing the self-correction of cognitive misappraisals. Thus, sudden reductions in obsessional fear and compulsive rituals occurring during ERP may be associated with changes in appraisals of intrusive thoughts.

ERP is also thought to promote tolerance of anxiety and fear (e.g., Craske et al., 2008, 2014, Jacoby & Abramowitz, 2016), and in this way it overlaps with acceptance-based therapies that foster willingness to experience unwanted thoughts and emotions such as obsessions, anxiety, and fear (e.g., Twohig et al., 2015). More specifically, experiential avoidance (EA) refers to one's unwillingness to tolerate unpleasant emotions, thoughts, or memories, and is thought to motivate maladaptive behaviors (e.g., compulsive rituals) that sustain obsessional distress (see Chawla & Ostafin, 2007). Sudden gains during ERP for OCD may therefore be related to reductions in EA, so we examined relationships between sudden gains and changes in both appraisals of intrusive thoughts and EA.

Data for the present study were collected as part of a two-site treatment study in which participants with OCD received ERP as the centerpiece of their treatment (Twohig et al., 2018). On the basis of previous research, we hypothesized that (a) approximately 50% of individuals would experience a sudden gain, (b) the rates of sudden gains would be highest among

participants with primary OCD symptoms related to contamination and lowest among those with primary OCD symptoms related to unacceptable thoughts, (c) individuals who experience a sudden gain would report significantly greater global and dimensional OCD symptom reduction at post-treatment and follow-up, even when accounting for magnitude of symptom change, and (d) individuals who experience a sudden gain would report significantly greater changes in interpretations of intrusive thoughts and experiential avoidance and at post-treatment and follow-up. We also explored whether demographic characteristics (i.e., gender, age, current medication treatment), OCD symptom severity, depressive symptom severity, and baseline cognitive measures would be predictors of sudden gains.

METHOD

Participants

Participants were 44 adults (28 female) between the ages of 18 and 56 (M = 27.19, SD = 8.22) who completed 16 sessions of manualized ERP treatment for OCD (see description further below). The sample was 80% (n = 35) Caucasian, 7% (n = 3) Hispanic, and 5% (n = 2) African American. All participants received a diagnosis of OCD according to the Structured Clinical Interview for DSM-IV (SCID-IV). Primary OCD symptom dimensions were determined using the Dimensional Obsessive-Compulsive Scale (see Measures section) and were represented as follows: contamination (n = 12), responsibility for harm (n = 15), unacceptable thoughts (n = 21), and symmetry (n = 5). The majority of participants had co-occurring DSM-IV diagnoses (52.3%; n = 23), with the highest frequencies of co-occurring mood disorders and anxiety disorders. 50% percent of the sample reported taking psychotropic medication during treatment (n = 22). Of these participants, the majority reported taking selective serotonin reuptake inhibitors (n = 13).

Measures

Participants completed the following measures of OCD symptom severity, depression, and psychological constructs.

Dimensional Obsessive-Compulsive Scale (DOCS; Abramowitz et al., 2010).

The 20-item self-report DOCS assesses the severity of four empirically validated OCD symptom dimensions: (a) contamination, (b) responsibility for harm and mistakes, (c) symmetry/ordering, and (d) unacceptable thoughts. Within each symptom dimension, five items

(rated 0 to 4) assess the following parameters of severity: (a) time occupied by obsessions and rituals, (b) avoidance behavior, (c) associated distress, (d) functional interference, and (e) difficulty disregarding the obsessions and refraining from the compulsions. The DOCS subscales have shown excellent reliability in clinical samples (α = .94–.96), and good convergent validity with other measures of OCD symptoms (Abramowitz et al., 2010). Participants completed the DOCS at pre-treatment, post-treatment, follow-up, and at the beginning of each treatment session.

The DOCS includes subscales that may not pertain to every participant's symptom presentation, and therefore could suppress the measure's sensitivity to treatment (Abramowitz et al., 2010). Therefore, in the present study, we used only the DOCS subscale with the highest score (i.e., most severe) at pre-treatment for each participant (called the "DOCS main" score). Once this primary OCD symptom dimension was determined, we computed DOCS main symptom scores at each session. In cases where two or more subscales tied for the highest score at pre-treatment (i.e., the patient had multiple "main" symptoms), subsequent DOCS main scores were computed as the mean of the corresponding subscale scores. This method of computing the most relevant symptom scores for each individual is consistent with Abramowitz and colleagues (2010).

Yale-Brown Obsessive Compulsive Scale (Y-BOCS; (Goodman et al., 1989a; Goodman et al., 1989b).

The Y-BOCS is a clinician-rated, 10-item structured interview assessing the global severity of obsessions and compulsions. The scale contains items assessing the following parameters of obsessions (items 1-5) and compulsions (items 6-10): (a) time, (b) interference, (c) distress, (d) resistance, and (e) degree of control associated with obsessions and compulsions. Each item is rated on a scale from 0 (*no symptoms*) to 4 (*extreme*), yielding total severity scores

that range from 0 to 40. The Y-BOCS has demonstrated good internal consistency, excellent inter-rater reliability, and good test-retest reliability (Goodman et al., 1989a; Goodman et al., 1989b). It is considered the gold standard measure of OCD symptom severity. Participants completed the Y-BOCS at pre-treatment, post-treatment, and follow-up.

Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996).

The BDI-II is a 21-item self-report scale that assesses the severity of affective, cognitive, motivational, vegetative, and psychomotor components of depression. Scores on the BDI-II range from 0 (no symptoms) to 63 (very severe symptoms). The BDI-II has excellent reliability and validity and is widely used in clinical research (Sprinkle et al., 2002; Steer, Ball, Ranieri, & Beck, 1999). Participants completed the BDI-II at pre-treatment, post-treatment, and follow-up.

Interpretation of Intrusions Inventory (III; Obsessive Compulsive Cognitions Working Group, 1997, 2001).

The III is a 31-item scale that assesses appraisals or interpretations of intrusive thoughts, images, or impulses. Respondents are provided with a definition of unwanted intrusions and illustrative examples, and are prompted to write two intrusive thoughts, images or impulses that they experienced recently. They then rate 31 statements as they pertain to intrusive thoughts like those recorded on the questionnaire using a scale from 0 ("I did not believe this idea at all") to 100 ("I was completely convinced this idea was true"). The instrument has demonstrated good validity, internal consistency, and reliability (Obsessive Compulsive Cognitions Working Group, 2005). Participants completed the III at pre-treatment, post-treatment, and follow-up.

Acceptance and Action Questionnaire-II (AAQ-II; Bond et al., 2011)

The AAQ-II is a 10-item scale that assesses EA. Participants rate their agreement with each of the seven statements (e.g., "I'm afraid of my feelings") on a 1 (*never true*) to 7 (*always true*) scale, such that higher scores indicate greater EA. The AAQ-II has demonstrated good

psychometric properties and good convergent, discriminant, and incremental validity (Bond et al., 2011; Fledderus, Oude Voshaar, Ten Klooster, & Bohlmeijer, 2012). Participants completed the AAQ at pre-treatment, post-treatment, and follow-up.

Obsessive Beliefs Questionnaire (OBQ; Obsessive Compulsive Cognitions Working Group, 2005).

The OBQ is a 44-item self-report instrument that measures dysfunctional beliefs (i.e., obsessive beliefs) hypothesized to underlie OCD symptoms. It contains three subscales: (a) threat overestimation and responsibility (OBQ-RT; 16 items), (b) perfectionism and need for certainty (OBQ-PC; 16 items), and (c) importance and control of thoughts (OBQ-ICT; 12 items). Participants rate items on a Likert scale ranging from 1 (Disagree very much) to 7 (Agree very much). The instrument has demonstrated good validity, internal consistency, and test-retest reliability (Obsessive Compulsive Cognitions Working Group, 2005). Participants completed the OBQ at pre-treatment, post-treatment, and follow-up.

Procedure

Data for this investigation were drawn from an OCD treatment study examining the effects of adding components of Acceptance and Commitment Therapy (ACT) to ERP. Twenty-three participants were treated with ERP alone and 21 received ERP that included components of ACT. The number of in-session hours dedicated to exposure therapy was equal in both conditions. Each participant completed 16 sessions of individualized treatment for OCD at one of two sites: University of North Carolina at Chapel Hill (UNC $n = 21$) and Utah State University (USU $n = 23$). Participants were randomly assigned to either the ERP treatment condition or the ERP+ACT treatment condition. Treatment was delivered by doctoral level therapists and advanced clinical psychology doctoral students who received training in the treatment protocol, adhered to detailed treatment manuals, and received supervision from doctoral-level clinical

psychologists with expertise in the treatment of OCD. Introductory sessions (1-3) for both treatment conditions included information gathering, psychoeducation, and treatment planning. Sessions 4-16 involved therapist supervised exposure with instructions to continue with similar exposure practice and refrain from rituals. Session 16 also included a discussion related to the end of treatment and how to prevent relapse. The centerpiece of both treatment conditions was ERP, yet in the ACT/ERP condition, metaphors drawn from ACT were included before, during and after each exposure trial to reinforce the concepts central to this approach (e.g., defusion from obsessional thoughts, acceptance of unwanted thoughts and anxiety, and the importance of ERP to one's values). Participants completed all study measures at the beginning and end of treatment. Data analyses from the parent trial indicated no group differences in treatment outcome between sites or treatment conditions (Twohig et al., 2018).

Definition of Sudden Gains

We used the criteria proposed by Tang and DeRubeis (1999) to identify sudden gains in our sample. These criteria require that the change between sessions is large (a) in absolute terms, (b) compared to severity before the gain, and (c) compared to fluctuations before and after the gain. These criteria were operationalized as follows:

Criterion A. As in previous research (e.g., Collins and Coles, 2017), we used the reliable change index (Jacobson & Truax, 1991) to identify gains that were large in absolute terms. We divided the difference scores between consecutive sessions by the standard error of the difference, and compared this value to 1.96. Values larger than 1.96 would be unlikely to occur due to chance alone, and thus satisfy Criterion A.

Criterion B. As defined by Tang & DeRubeis (1999), the difference scores between consecutive sessions must represent at least 25% of the pre-gain score to satisfy Criterion B

Criterion C. To determine if a gain is large relative to fluctuations before and after the gain, we conducted independent sample t-tests to compare the three before-gain and after-gain DOCS main scores. Replicating the sudden gains calculations of Hardy and colleagues (2005), we used cutoffs to determine whether or not difference scores constituted sudden gains. Gains between two consecutive sessions met criterion C if $t \geq 2.50$ ($t \geq 3.00$ if only two pre-gain or after-gain scores were available). If only one pre-gain score was available, sudden gains were not identified. Therefore, gains occurring immediately after session 1 were not included in analyses.

Data Analysis

We tested our first hypothesis that 50% of individuals would experience a sudden gain by measuring the occurrence of sudden gains using the criteria outlined above. We then tested our second hypothesis that sudden gains would be most prevalent for participants with primary contamination symptoms and least prevalent for those with primary unacceptable thoughts symptoms by comparing the rates of sudden gains across DOCS main symptom groups.

We tested our third hypothesis that individuals who experience a sudden gain would report significantly greater overall OCD symptom reduction at post-treatment in both the full sample and a sample of matched pairs. The sample of matched pairs was created to control for magnitude of symptom change. We matched each participant who experienced a sudden gain with a participant who did not, based on comparable symptoms at the first treatment session (i.e., less than or equal to a 1-point difference on DOCS main symptoms) and at the post-gain session (i.e., less than or equal to a 3-point difference on DOCS main symptoms). We then conducted mixed ANOVA tests using both the full sample and the sample of matched pairs. For all tests, independent variables were time (2 level within subject variable: pre-treatment vs. post-treatment) and presence of sudden gains (2 level between subject variable: sudden gain vs. no

sudden gain). The dependent variables were DOCS main and Y-BOCS total score. We examined these relationships at follow-up by conducting mixed ANOVAs with time (post-treatment vs. follow-up) as the independent variable.

We tested our fourth hypothesis that individuals who experience a sudden gain would report significantly greater reductions in experiential avoidance and intrusive thoughts at post-treatment by conducting mixed ANOVA tests with time and presence of sudden gains as independent variables and the AAQ and III as dependent variables. Finally, we explored predictors of sudden gains by conducting individual logistic regressions to test whether patient characteristics (gender, age, current medication treatment), pre-treatment symptom severity (DOCS main, YBOCS, BDI-II), and/or pre-treatment cognitions (OBQ, III, AAQ) predicted the occurrence of a sudden gain.

RESULTS

Occurrence of sudden gains

Using the criteria derived by Tang and DeRubeis (1999) outlined above, we identified 12 participants who experienced a sudden gain, representing approximately 27% of the sample. The average sudden gain magnitude was 5.58 points on the DOCS main symptom subscale (*SD* = 2.12). The average total symptom reduction for the full sample from pre- to post-treatment was 7.02 points on the DOCS main (*SD* = 3.76), and the average total symptom reduction for participants who experienced a sudden gain was 9.25 points on the DOCS main (*SD* = 3.91). Thus, for participants who experienced sudden gains, such gains accounted for an average of 60.3% of total symptom reduction. Regarding the first sudden gain experienced by these participants, most occurred after session 3 (*n* = 3), after session 4 (*n* = 2), after session 6 (*n* = 3), and after session 10 (*n* = 2).

Sudden gains by OCD symptom dimension

Of the 12 participants with primary contamination symptoms, 6 (12%) experienced a sudden gain. Of the 15 participants with primary OCD symptoms related to responsibility for harm, 2 (13.3%) experienced a sudden gain. Of the 21 participants with primary OCD symptoms related to unacceptable thoughts, 5 (23.8%) experienced a sudden gain. Finally, of the 5 participants with primary OCD symptoms related to symmetry, 1 (20%) experienced a sudden gain.[1]

[1] Although it would be desirable to conduct a chi-square test to statistically compare the frequencies of sudden gains across OCD symptom presentations, the data are non-independent as some participants were part of multiple DOCS main groups. This created a situation in which there were many cells with few observations, precluding chi-square analyses.

Sudden gains and outcome in the full sample

Pre-treatment, post-treatment, and follow-up scores for the full sample by sudden gain status are presented in Table 1. There were no significant pre-treatment differences in DOCS main or Y-BOCS scores between participants who experienced sudden gains and those who did not (all p's > .05). As expected, a significant main effect of time (pre- to post-treatment) was observed for DOCS main symptom scores ($F(1,41) = 165.20$, $p < .001$, $\eta_p^2 = .80$) and Y-BOCS scores ($F(1,42) = 250.05$, $p < .001$, $\eta_p^2 = .86$). This effect was maintained at follow-up for both DOCS main symptom scores ($F(1,36) = 128.62$, $p < .001$, $\eta_p^2 = .78$) and Y-BOCS scores ($F(1,41) = 189.04$, $p < .001$, $\eta_p^2 = .82$), indicating that participants experienced substantial improvement in OCD symptoms that were maintained at follow-up. We also found a significant time by sudden gain interaction for DOCS main symptom scores, such that participants who experienced a sudden gain had greater average symptom reduction from pre- to post-treatment, than did those without a sudden gain ($F(1,41) = 6.64$, $p = .014$, $\eta_p^2 = .14$). However, these significant relationships did not persist at the six-month follow-up assessment ($F(2,72) = 3.10$, $p > .05$, $\eta_p^2 = .08$). Contrary to our hypotheses, there were no significant time by sudden gain interactions as assessed by the Y-BOCS, AAQ-II, or III scores (all p's > .05).

Sudden gains and outcome in the sample of matched pairs

Each of the 12 participants who experienced a sudden gain was matched with a participant who did not experience a sudden gain, but who achieved comparable reduction in DOCS main scores between the first treatment session and at the post-gain session. Pre-treatment, post-treatment, and follow-up scores for the sample of matched pairs are presented in Table 2. As expected, in this sample of matched pairs ($n = 24$), a significant main effect of time (pre- to post-treatment) was observed for DOCS main ($F(1,22) = 118.27$, $p < .001$, $\eta_p^2 = .84$) and

Y-BOCS scores ($F(1,22) = 164.69$, $p < .001$, $\eta_p^2 = .88$). This effect was maintained at follow-up for both DOCS main ($F(1,20) = 74.18$, $p < .001$, $\eta_p^2 = .79$) and Y-BOCS scores ($F(1,22) = 81.97$, $p < .001$, $\eta_p^2 = .79$). However, there was no significant time by sudden gain interaction for DOCS main scores between pre- and post-treatment ($F(1,22) = .762$, $p > .05$, $\eta_p^2 = .03$), or when follow-up was included in the model ($F(2,40) = .980$, $p > .05$, $\eta_p^2 = .05$). There were also no significant time by sudden gain interactions for Y-BOCS, AAQ-II, or III scores in this sample (all p's > .05).

Pre-treatment predictors of sudden gains

Current medication was the only significant pre-treatment predictor of sudden gains, such that medication use was associated with an increased likelihood of experiencing a sudden gain (OR = 6.23, $p < .05$). Gender, age, DOCS main scores, Y-BOCS scores, depressive symptom severity, obsessive beliefs, interpretations of intrusive thoughts, and experiential avoidance at baseline were not significant predictors of sudden gains (all p's > .05).

DISCUSSION

Numerous studies document sudden gains during treatment of depression and anxiety disorders, and some have found that sudden gains are associated with enhanced treatment outcome. The only two studies that have examined this relationship during OCD treatment, however, reported conflicting findings. Thus, the purpose of the present study was to shed additional light on sudden gains during ERP for OCD, and to address important conceptual and methodological limitations of the existing literature. Specifically, we examined the frequency of sudden gains, as well as their relationship to symptom presentation and treatment outcome at both post-treatment and follow-up, and controlled for the magnitude of the gain. Moreover, we examined whether sudden gains were related to changes in psychological constructs (e.g., interpretations of intrusive thoughts) involved in the maintenance of OCD symptoms.

In partial support of our first hypothesis we found sudden gains in our sample, yet only 27% of participants experienced a sudden gain. These gains occurred throughout treatment, suggesting that there was no consistent temporal pattern of sudden gains across individuals receiving ERP for OCD. The majority of sudden gains occurred after sessions 3, 4, 6, and 10. The rate of sudden gains in our data was notably lower than rates observed in previous studies of sudden gains during OCD treatment (i.e., Aderka et al., 2012; Collins & Coles, 2017). A closer look at the relationship between symptom presentation and sudden gains revealed that, consistent with our second hypothesis, the highest rate of sudden gains was achieved among participants with primary contamination symptoms. Consideration of this relationship, along with the underrepresentation of contamination symptoms in our sample, provides a possible explanation

for the difference in overall rates of sudden gains between the present study and previous investigations. Specifically, only 27% of our sample had primary contamination symptoms, which is far lower than what would be expected given meta-analytic findings that contamination symptoms predominate among participants in OCD treatment outcome studies (e.g., 48% in Ball et al., 1996). Thus, although not reported, both previous studies may have had more participants with primary contamination symptoms, which could account for the higher overall rates of sudden gains.

The sudden gains experienced by individuals with primary contamination symptoms may be explained by research suggesting that contamination-related symptoms are particularly responsive to ERP (Abramowitz, Franklin, Schwartz, & Furr, 2003). Symptom decreases may occur more rapidly for individuals with contamination obsessions and cleaning rituals because exposure exercises that target threat overestimations related to contamination (e.g., I will get sick if I make contact with germs) are generally straightforward (e.g., touch a doorknob). Furthermore, unlike mental rituals used to neutralize other types of obsessions, washing and cleaning rituals in response to contamination obsessions are often clearly observable, and therefore targetable, by both client and therapist.

We expected to find the lowest rates of sudden gains among participants whose symptoms related to unacceptable thoughts accompanied by mental rituals. Indeed, such individuals are most likely to have attenuated outcomes with ERP. We instead found that rates were lowest among individuals with primary symptoms related to responsibility for harm and the urge to check or seek reassurance. Symptoms related to both unacceptable thoughts and responsibility for harm can be challenging to target with ERP, as cognitions associated with these symptom presentations relate to the significance of thoughts and the importance of

controlling them. Perhaps changes in these cognitions (i.e., learning that intrusive thoughts are safe and tolerable, and don't need to be controlled) occur more gradually during exposure relative to changes in overestimates of threat related to contamination. In addition, treatment of such symptoms often focuses on building uncertainty tolerance (Abramowitz & Jacoby, 2015), and this process may be slower than correcting threat overestimations related to contamination. Differences in treatment strategies across symptom presentations may help explain different rates of sudden gains between dimensions.

Consistent with our third hypothesis and the findings of Aderka and colleagues (2012), individuals who experienced a sudden gain had larger symptom reductions than those who did not. However, this relationship was not maintained at follow-up, suggesting that the relevance of sudden gains to symptom reduction lessens over time after the completion of a course of treatment. Interestingly, however, when participants who experienced a sudden gain were matched with those who experienced a gradual gain of similar magnitude, sudden gains were not uniquely associated with treatment outcome at post-treatment or follow-up. This finding suggests that it is not the *suddenness* of the gain that is associated with improved outcome, but rather the magnitude of the gain. It is also possible that the reduced sample size for this analysis limited the power to detect a statistically significant effect, although the effect sizes we found ($\eta_p^2 = .03$ at post-treatment and $\eta_p^2 = .05$ at follow-up) suggest that this was not the case. It is notable that our findings differ from those of Greenfield, Gunthert, and Haaga (2011), who found significant differences between "sudden gainers" and "gradual gainers" in a diagnostically heterogeneous outpatient sample. However, important methodological differences between our study and theirs (e.g., diagnostic status, treatment setting) may account for this difference.

In contrast with our fourth hypothesis, there were no significant relationships between the

experience of a sudden gain and changes in interpretations of intrusive thoughts or experiential avoidance during treatment. Thus, although associated with overall decreases in obsessions and compulsions at post-treatment, rapid symptom reduction during ERP was not associated with changes in psychological constructs that have been shown to maintain these symptoms. These inconsistencies with previous findings may be explained by the specific constructs of interest in this study, which differ from the cognitive factors that were associated with sudden gains in previous studies. It is important to note that these findings do not rule out the possibility that sudden gains were associated with cognitive-behavioral changes during and between individual sessions.

Regarding our exploratory analyses, we found that medication status was the only pre-treatment predictor of sudden gains.[2] Psychotropic medication may support engagement with exposure exercises, thus promoting rapid and substantial improvement during ERP. Indeed, some studies suggest that medication augments behavioral interventions for OCD (e.g., Hohagen et al., 1998). Other studies, however, have found that the addition of medications does not improve outcomes relative to ERP alone (e.g., Foa et al., 2005; for a review see van Balkom et al., 1994). It is also possible that placebo or context effects associated with medication (i.e., awareness that one is using an agent thought to be helpful) contributed to better short-term (although not necessarily better long-term; e.g., Craske et al., 2008) outcomes. This is especially plausible given that medication was not randomly assigned in this study. Previous studies have not found relationships between medication use and sudden gains (e.g. Collins & Coles, 2017; Clerkin et al., 2008), so additional research is necessary to explore medication as a potential moderator of sudden gains.

[2] Several drug classes were represented in our sample, including selective serotonin reuptake inhibitors, anxiolytics, and antipsychotics, precluding class-specific conclusions about the relationship between medication status and sudden gains.

Consistent with both Aderka and colleagues (2012) and Collins and Coles (2017), gender, age, dimensional and global OCD symptom severity, depressive symptom severity, obsessive beliefs, experiential avoidance, and intrusive thoughts at baseline did not predict the occurrence of a sudden gain. Collins and Coles suggested that although one might expect a positive relationship between pre-treatment symptom severity and the likelihood of a sudden gain due to regression to the mean, individuals with more severe symptoms may experience change more gradually. It is also possible that some true predictors of sudden gains were not measured in the present study (e.g., motivation, insight into symptoms, treatment expectancy, social support).

Sudden gains during ERP for OCD, and their use as prognostic indicators, are of interest to investigators who study OCD treatment with the hope of improving outcomes. In light of our findings, however, we recommend caution when considering the significance of this phenomenon. First, sudden gains do not appear to be reliable predictors of ERP outcomes for specific symptom dimensions or for OCD symptoms globally. Although sudden gains predicted improvement in participants' primary obsessions and compulsions at post-treatment, this relationship did not persist at follow-up. Moreover, when we controlled for the magnitude of the gain, the occurrence of a sudden gain was no better at predicting outcome than was a gradual gain of a similar magnitude. Furthermore, sudden gains were not associated with psychological constructs that have consistently demonstrated relevance to OCD symptom reduction (i.e. experiential avoidance and interpretations of intrusive thoughts). Thus, despite encouraging findings from previous research on sudden gains, our results indicate that alternative outcome predictors may hold more promise for improving our understanding of symptom change during ERP for OCD.

The present study has limitations that should be considered when drawing conclusions.

Several therapists (mostly graduate-level) delivered ERP to participants, and sessions were not scored for treatment integrity. While this study design improved the generalizability of our results to typical community outpatient settings with multiple clinicians, it may have compromised internal validity due to differences in therapeutic styles. Conversely, several design decisions prioritized internal over external validity, as is typical of randomized controlled trials. For example, individuals who had severe co-occurring disorders or previous experience with CBT for OCD were excluded from participating, therapists used detailed treatment manuals, and every case was supervised by a doctoral-level clinician. These conditions likely do not reflect the level of diagnostic heterogeneity or treatment delivery in typical treatment settings. Additional research on sudden gains is warranted in settings where there is greater diversity of patients and therapists. Finally, our sample size may have limited our power to detect effects, particularly in our reduced sample of matched pairs.

Despite these limitations, the present study adds to the existing literature on sudden gains during OCD treatment. Although sudden gains have been repeatedly linked to treatment outcome in depression and anxiety treatment, the present study casts doubt on the relevance of sudden gains to long-term outcome in OCD treatment. We agree with the sentiment expressed by Collins and Coles (2017) that the relationship between sudden gains and outcome during psychological treatment is an atheoretical investigation of an observed clinical phenomenon. Future research that empirically investigates theory-driven relationships may lead to more precise identification of treatment mechanisms, and ultimately contribute to improving outcomes for individuals with OCD.

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Table 1

Pre-treatment, post-treatment, and follow-up mean scores (SDs) for participants with and without a sudden gain.

	Sudden Gain			No Sudden Gain		
	Pre-treatment	Post-treatment	Follow-up	Pre-treatment	Post-treatment	Follow-up
DOCS main	14.42 (2.84)	5.17 (3.32)	5.25 (3.81)	13.19 (2.66)	7.13 (3.09)	6.28 (3.79)
Y-BOCS	24.42 (3.94)	9.75 (5.24)	9.17 (6.41)	25.25 (4.27)	11.94 (4.73)	12.35 (7.17)
III	1392.50 (496.79)	632.50 (540.47)	671.00 (765.60)	1405.31 (562.08)	996.44 (685.27)	758.28 (651.56)
AAQ-II	30.25 (9.19)	21.08 (8.41)	19.00 (8.56)	29.88 (8.07)	25.13 (8.74)	23.14 (8.48)
BDI-II	16.58 (10.72)	5.83 (6.15)	7.50 (5.76)	16.41 (10.54)	8.41 (6.83)	8.83 (8.07)
OBQ	196.58 (48.16)	121.67 (36.51)	116.86 (28.49)	198.28 (48.20)	152.31 (51.76)	136.37 (53.53)

Table 2

Pre-treatment, post-treatment, and follow-up mean scores (SDs) for the sample of matched pairs.

	Sudden Gain			No Sudden Gain		
	Pre-treatment	Post-treatment	Follow-up	Pre-treatment	Post-treatment	Follow-up
DOCS main	14.42 (2.84)	5.17 (3.32)	5.25 (3.81)	13.83 (3.24)	5.96 (2.54)	6.88 (4.10)
Y-BOCS	24.42 (3.94)	9.75 (5.24)	9.17 (6.41)	25.83 (3.56)	11.83 (3.49)	14.42 (8.75)
III	1392.50 (496.79)	632.50 (540.47)	671.00 (765.60)	1557.50 (479.55)	723.33 (511.26)	856.67 (759.88)
AAQ-II	30.25 (9.19)	21.08 (8.41)	19.00 (8.56)	32.42 (6.61)	23.58 (7.50)	24.50 (9.89)
BDI-II	16.58 (10.72)	5.83 (6.15)	7.50 (5.76)	17.58 (10.12)	8.00 (6.37)	10.17 (8.77)
OBQ	196.58 (48.16)	121.67 (36.51)	116.86 (28.49)	199.20 (71.58)	132.00 (56.46)	138.20 (62.31)